suggest either yersiniosis or post-*Salmonella* or post-*Shigella* arthritis. Failure to show a significant titer at four and eight weeks after onset underscores the importance of obtaining the first serologic specimen early in the course of illness. (Serologic studies reported here were carried out by the Microbial Diseases Laboratory of the State of California, Department of Health, 2151 Berkeley Way, Berkeley, CA 94704.)

Table 1 presents similarities and differences in clinical and laboratory data for the four cases reported here.

Conclusion

The four cases reported show that reactive arthritis following enteritis due to *Y enterocolitica* may occur in persons residing in Northern California, a syndrome previously reported most frequently in Scandinavia. Because reactive arthritis is known to occur with greatest frequency in persons who are HLA-B27 positive, a test for this antigen should be done in patients in whom arthritis in association with a febrile or diarrheal disease has developed, particularly if *Yersinia, Salmonella* or *Shigella* infection is suspected or proved. At this time the prognosis for these patients is not known. It had been thought that this syndrome was always self-limited, but Calin and Fries[3] have found some patients with prolonged and persistent arthritis following diarrheal disease due to *Shigella.*

REFERENCES

1. Ahvonen P, Sievers K, Aho K: Arthritis associated with *Yersinia enterocolitica* infection. Acta Rheumatol Scand 15:232-253, 1969
2. Warren CPW: Arthritis associated with *Salmonella* infections. Ann Rheumat Dis 29:483-487, 1970
3. Calin A, Fries JF: An "experimental" epidemic of Reiter's syndrome revisited—Follow-up evidence on genetic and environmental factors. Ann Intern Med 84:564-566, 1976
4. Aho K, Ahvonen P, Lassus A, et al: HL-A27 in reactive arthritis—A study of Yersinia-arthritis and Reiter's disease. Arthritis Rheum 17:521-526, 1974
5. Friis J, Svejgaard A: Salmonella arthritis and HL-A27 (Letter). Lancet 1:1350, 1974
6. Chalmers A, Kaprove RE, Reynolds WJ, et al: Postdiarrheal arthropathy of *Yersinia pseudotuberculosis.* Can Med Assoc J 118: 515-516, 1978
7. Gutman LT, et al: An inter-familial outbreak of *Yersinia enterocolitica* enteritis. N Engl J Med 288:1372-1377, 1973
8. Ford DK, Henderson E, Price GE, et al: Yersinia-related arthritis in the Pacific Northwest. Arthritis Rheum 20:1226-1230, 1977
9. Jacobs JJ: *Yersinia enterocolitica* arthritis. Pediatrics 55:236-238, 1975
10. Sonnenwirth AC, Weaver RE: *Yersinia enterocolitica* (Letter). N Engl J Med 283:1468, 1970
11. Kohl S, Jacobson JA, Nahmias A: *Yersinia enterocolitica* infections in children. J Pediatr 89:77-79, 1976
12. Marks MI, Pai CH, Lafleur L, et al: *Yersinia enterocolitica* gastroenteritis in children (Abstract). Pediatr Res 12:496, 1978
13. Sonnenwirth AC: Yersinia, *In* Lennette EC, Spaulding EH, Truant JP (Eds): Manual of Clinical Microbiology, 2nd Ed. Washington, DC, Society of Microbiology, 1974, pp 222-230
14. Leino R, Kalliomaki JL: Yersiniosis as an internal disease. Ann Intern Med 81:458-461, 1974
15. Ahvonen P: Human yersiniosis in Finland—I. Bacteriology and serology. Ann Clin Res 4:30-33, 1972

Refer to: Jung RC, Aaronson J: Death following inhalation of mercury vapor at home. West J Med 132:539-543, Jun 1980

Death Following Inhalation of Mercury Vapor at Home

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THE INHALATION of toxic fumes and metallic vapors is a fairly common occurrence in industry. There are ample accounts describing the effects from the inhalation of vaporized mercury on the respiratory tract and other organ systems.[1-5] Ramazzini[6] in 1940 gave one of the earliest descriptions of the consequences of such exposure. Studies indicate that members of the dental profession and workers in the fur or felt hat-making industry have been at greatest risk for exposure to mercury.[2] Other industrial exposures have occurred primarily when metallic mercury has been accidentally vaporized in a high-temperature environment.[3-5]

Fortunately, deaths among adults exposed in industry have been very rare.[4] In contrast, accidental exposure to mercury has resulted in deaths among children and has caused chronic disability in adults. In the following report we discuss what appears to be the first death of an adult exposed to mercury vapor in the home. We have included hemodynamic measurements, which have not been described previously in these patients.

Report of a Case

A 53-year-old man, suffering from severe respiratory distress, was transferred to the Los Angeles County/University of Southern California Medical Center for specialized renal and respiratory care. He had been in good health until approximately two weeks before admission. At that time, while the patient was working with elemental mercury in an ore distillation process on his kitchen stove, chills, fever, dyspnea, headache,

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Submitted July 16, 1979.

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ABBREVIATIONS USED IN TEXT

CPAP = continuous positive airway pressure
ECMO = extracorporeal membrane oxygenator
PEEP = positive end-expiratory pressure

red spots in front of his eyes, diarrhea and abdominal cramps developed. A few hours later he complained of partial blindness; however, all the symptoms gradually disappeared over the next two days. Six days before admission he again heated some ore and mercury mixture over his kitchen stove for about 20 minutes. Shortly thereafter he noted the room becoming dark, and the red spots in his eyes reappeared. He became weak, vomited once and then experienced headache, chills, fever and diarrhea. These symptoms continued for the next four days. He then became rapidly and increasingly short of breath, and was admitted to hospital the next day. At that time he was found to be cyanotic, in acute respiratory distress, and having severe abdominal cramps. A roentgenogram of the chest showed bilateral lung infiltrates consistent with pulmonary edema. Because he had had a myocardial infarction within the past year, a balloon-tipped floating catheter was inserted into the pulmonary artery. The pulmonary artery and wedge pressures were reported to be normal. A tracheostomy was done and his respirations were mechanically assisted. The level of mercury in his urine was 5 mg per dl. Because charcoal hemodialysis was being considered he was transferred to the medical center. On arrival, the patient's temperature was 37.1°C (98.8°F); blood pressure, 130/100 mm of mercury; respirations, 18 per minute when assisted by a ventilator, and pulse, 76 beats per minute. He was alert, oriented and in no apparent distress. At this time, he was not cyanotic. Inspiratory rales were present in both lung bases. There were hypoactive bowel sounds. Results of the neurological examination showed no abnormalities. The initial pulmonary artery pressure was 34/20 mm of mercury, with a mean wedge pressure of 10 mm of mercury. A roentgenogram of the chest (Figure 1) showed extensive bilateral pulmonary infiltrates but no evidence of cardiomegaly. An analysis of arterial blood gases, while the patient breathed 70 percent oxygen, with a positive end-expiratory pressure (PEEP) of 12 cm of water, showed oxygen pressure (P_{O_2}) 73 torr; pH 7.42; carbon

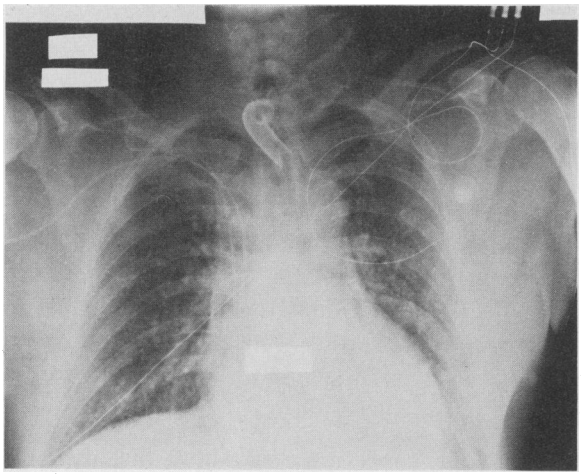

Figure 1.—A roentgenogram of the patient's chest showing extensive bilateral pulmonary infiltrates but no evidence of cardiomegaly.

dioxide pressure (P_{CO_2}) 35 torr, and bicarbonate 22 mEq per liter. Other laboratory data were as follows: hematocrit 48 percent; hemoglobin 16.6 grams per dl; leukocyte count 20,000 per cu mm, with 89 percent polymorphonuclear cells, 5 percent bands, 5 percent lymphocytes and 1 percent monocytes; occasional erythrocytes contained inclusion bodies resembling siderocytes. An analysis of the urine showed a specific gravity of 1.025 and pH of 5.5; proteinuria (1+); glucosuria (1+); and one to two pus cells, zero to one erythrocyte, no casts and few bacteria per low-power field. Blood chemistry measurements were within normal limits except for the following: sodium 131 and chloride 94 mEq per liter; and glucose 172, urea nitrogen 45, uric acid 9.6 and creatinine 1.5 mg per dl. Lactic dehydrogenase measured 2,400 units. The urinary concentration of mercury was 3,700 μg per liter (normal, up to 20 μg per liter). Other urine measurements were sodium 40 and potassium 54 mEq per liter; osmolality 792 mOsm per liter, and creatinine 102 mg per kg of body weight per 24 hours. Findings on an electrocardiogram showed an old inferior myocardial infarction.

Following intravenous administration of 500 ml of 5 percent dextrose in water over four hours the patient's pulmonary artery pressure rose to 40/24 mm of mercury, with the mean wedge also rising to 22 mm of mercury. His urinary output was 100 ml per hour. The intravenous infusion rate was lowered, resulting in a lowering of the wedge pressure. During the next four hours his respiratory support was converted to continu-

ous positive airway pressure (CPAP) with a fraction of inspired oxygen (FIO_2) of 0.5. He was given a course of dimercaprol (BAL in Oil). By the seventh hospital day the urine mercury level was 660 μg per liter. The urea nitrogen and creatinine levels fell to within normal range and he continued to excrete adequate urine. With continued modest fluid restriction his serum sodium and chloride concentrations returned toward normal. On the ninth day he suddenly complained of pain in the right side of his chest, and acute tachypnea and respiratory distress developed. A roentgenogram of the chest showed a massive pneumothorax and empyema. Chest tube drainage was instituted. *Pseudomonas aeruginosa* and coagulase-positive *Staphylococcus aureus* were isolated from the tracheal aspirate. He was given tobramycin and methicillin. Because it became increasingly difficult to maintain adequate oxygenation with mechanical ventilation, the patient was transferred to another facility for extracorporeal membrane oxygenator (ECMO) support. During the next 24 hours, before ECMO could be carried out, there seemed to be improvement, which continued for the next 12 days without ECMO. However, the pneumothorax with bronchopleural fistula persisted, and tracheomalacia developed as well. The patient's condition then worsened: he acquired nosocomial pneumonia and died during the third week of this hospital stay.

Discussion

This patient undoubtedly died from nosocomial pneumonia, but, unquestionably, a strong contributing factor was his two brief but severe exposures to mercury vapor. His symptoms and signs during both episodes were similar to those which have been previously reported. The delayed onset of respiratory distress that followed the initial symptoms is also characteristic. The classic description by Matthes[7] of the symptoms experienced by a mother and her three children exposed overnight to fumes emanating from a gas stove, which had been freshly painted with paint that contained mercury, has been repeated in other accounts of accidental exposure. Hallee[8] reported a family of five who were exposed to mercury vapors when the father used the metal to extract gold from gold sand over a kitchen stove. Their presenting symptoms and signs matched those seen in our patient. His exposure history was virtually identical except that the dose was probably higher because his exposure occurred on two occasions over a short time.

Although the dose of exposure is best determined by measuring the concentration of mercury in whole blood,[9] most accounts of inhalation injury report concentrations of the substance in urine. Because urine levels are both protracted and irregular,[10] it does not accurately reflect the high levels achieved soon after exposure. When one considers that normal levels of mercury in the urine are 0 to 20 μg per liter, then our patient's levels were certainly in the high toxic range. Urine levels in patients with similar symptoms and pulmonary alterations have been reported to be between 540 μg and 2,100 μg per liter per 24 hours.[11]

Although the dose of mercury usually inhaled by most persons is not lethal, deaths have occurred. Campbell[12] reported the first known death, that of a 4-month-old infant who had been exposed to a teaspoon of mercury vaporized over a hot kitchen stove. At autopsy, the child had pulmonary edema, renal tubular damage and mucosal necrosis of the stomach and duodenum. These organ systems as well as the brain are considered to be major sites of deposition of mercury after it is absorbed through the lungs and into the blood. In the detailed postmortem examination of the three children in the report by Matthes,[7] there was erosive bronchitis and bronchiolitis, with severe interstitial pneumonitis and hyaline membrane. There was also alternating alveolar collapse or emphysema and pneumothorax. Only minor changes were seen in the liver and kidney. During the early course of our patient's illness a pneumothorax developed. Although possible, the mechanical support of his breathing (CPAP, 8 to 10 cm of water) probably was less at fault than were the morphological changes caused by his underlying illness, which made the lung more susceptible to barotrauma.

The radiological appearance of the lungs in our patient is consistent with that described previously.[4,8,13] These all reflect the development of acute noncardiogenic pulmonary edema. The pulmonary artery pressure measurements support this diagnosis in our patient. Even though he had a history of previous heart disease, the mean wedge pressures were persistently normal during the early hours of his acute illness. Later, his wedge pressure rose after fluid administration, which may then have reflected some myocardial malfunction.

Teng[14] described the radiological abnormalities seen in the children reported by Matthes[7] and correlated them with changes seen at autopsy. There were substantially depressed diaphragms, ill-defined areas of increased radiodensity due to pulmonary edema, diffuse alveolar and interstitial emphysema, and pneumothorax.

The distribution of inhaled mercury vapor has been extensively studied. Berlin[15] compared the concentration of mercury deposited in the brain of mice given equivalent doses of either the inhaled vapor or mercuric nitrate by intravenous injection. Uptake was ten times greater by the former route. Kudzk[16] allowed four human subjects to inhale mercury vapor and measured the inspired and expired concentrations. From his results he concluded that the absorption of mercury was almost complete and was independent of the dose inhaled. Teisinger[10] exposed a group of volunteers to 100 mg per cu mm of mercury vapor for seven hours. About 76 percent of the mercury was retained in the mouth and nose. Therefore, the actual amount absorbed could not be accurately estimated. On the other hand, Berlin and co-workers[17] reported that when guinea pigs inhaled mercury vapor the highest concentrations of the substance were found in the periphery of the lung, and that most of the mercury was quickly transferred to the blood. Only a small fraction remained in the lung tissue, and this was slowly distributed to the rest of the body. Using radioisotope techniques, Hayes[18] found similar results in rats. Matthes[7] measured mercury concentrations in tissue from the livers, lungs and kidneys of the children who had died. The highest concentrations were found in the kidneys (60 to 79.1 mg per kg of body weight) whereas the lungs had the lowest amounts (6.3 to 9.3 mg per kg of body weight). One must conclude that the inhalation of mercury vapor, even in small concentrations, exposes a person to a very highly toxic substance, which can lead to serious or lethal complications.

Had our patient survived, one could speculate on the long-term outcome of his exposure. There are only two accounts which deal with the late effects on persons who have inhaled mercury vapor. In the first report, Hallee[8] noted that the father (who had received the most serious exposure and who had persistent abnormal pulmonary function) had evidence of diffuse alveolar fibrosis on a lung biopsy done five months after exposure. He and his family had been exposed to no more than 30 ml of mercury vapor. The second account[4] describes six workers who were exposed to mercury vapor from a broken thermostat for approximately eight hours. All were symptomatic, and roentgenograms of the chest showed abnormalities in three. They all complained of persistent impotency, fatigue, and vague personality disturbances that continued for as long as eight years after the accident.

Treatment for this problem, beyond symptomatic support, is disappointing. Immediate support with CPAP or, if the victim is hypercapneic, with mechanical ventilation with PEEP is appropriate and indicated. Our patient initially responded to this form of therapy. The only therapy suggested to promote the elimination of mercury from the body is dimercaprol. However, it has not been shown to be highly successful. The recommended dosage is 3 mg per kg of body weight every six hours for three to four days.[11] Charcoal perfusion hemodialysis, although initially considered by us, may be inappropriate and probably is not an efficient method of eliminating mercury bound to the tissues. Finally, all efforts should be made to prevent the development of a nosocomial lung infection, which, unfortunately, occurred in our patient.

Clearly, the best way to prevent such a mishap is to avoid exposure. However, even the most well-intentioned, but unknowledgeable, persons can become exposed to the hazards of mercury vapors.[13,19] It then becomes essential for physicians to understand the urgency for taking optimal therapeutic measures so that the survival of such patients may be assured.

Summary

On two separate occasions, a 53-year-old man inhaled mercury vapor while heating the metal over a kitchen stove. Noncardiogenic pulmonary edema developed (diagnosis supported by hemodynamic measurements) from which he temporarily recovered. However, he died subsequently from nosocomial pneumonia. This may be the first recorded death of an adult in a nonindustrial setting.

REFERENCES

1. Tennant R, Johnstone HJ, Wells JB: Acute bilateral pneumonitis associated with the inhalation of mercury vapor. Conn Med 25:106-109, 1961

2. Neal PA, Jones RR: Chronic mercurialism in the hatters' fur cutting industry. JAMA 110:337-343, Jan 1938

3. Seaton A, Bishop CM: Acute mercury pneumonitis. Br J Indust Med 35:258-265, Jul 1978

4. McFarland RB, Reigel H: Chronic mercury poisoning from single brief exposure. J Occup Med 20:532-534, Aug 1978

5. Milne J, Christopher H, DeSilva P: Acute mercurial pneumonitis. Br J Indust Med 27:334-338, Oct 1970

6. Ramazzini B: De Morbis Artificum Diatriba (1713), Wright WC (trans). Chicago, U of Chicago Press, 1940

7. Matthes FT, Kirschner R, Yow MD, et al: Acute poisoning associated with inhalation of mercury vapor. Pediatrics 22:675-688, Oct 1958

8. Hallee JT: Diffuse lung disease caused by inhalation of mercury vapor. Am Rev Resp Dis 99:430-436, Mar 1969

9. Berlin M, Fazackerley J, Nordberg G: The uptake of mercury in the brains of mammals exposed to mercury vapor and mercuric salts. Arch Environ Health 18:719-729, May 1969

10. Teisinger J, Fiserova-Bergerova V: Pulmonary retention and excretion of mercury vapors in man. Indust Med Surg 34:580-584, Jul 1965

11. Haddad JK, Stenberg E Jr: Bronchitis due to acute mercury inhalation. Am Rev Resp Dis 88:543-545, Oct 1963

12. Campbell JS: Acute mercurial poisoning by inhalation of metallic vapor in an infant. Canad Med Assn J 58:72-75, Jan 1948

13. Burke WJ, Quagliana JM: Acute inhalation mercury intoxication. J Occup Med 5:157-160, Feb 1963

14. Teng CT, Berman JC: Acute mercury vapor poisoning. Radiology 73:354-359, Sep 1959

15. Berlin M, Johannson LG: Mercury in mouse brain after inhalation of mercury vapor and after intravenous injection of mercury salt. Nature 204:85-86, Oct 1964

16. Kudzk FN: Absorption of mercury vapor from the respiratory tract in man. Acta Pharmacol Toxicol 23:250-262, Nov 1965

17. Berlin M, Nordberg GF, Serevins F: On the site and mechanism of mercury vapor resorption in the lung, *In* Nordberg GF (Ed): Effects and Dose-Response Relationships of Toxic Metals. Amsterdam, Elsevier, 1976

18. Hayes AD, Rothstein A: The metabolism of inhaled mercury vapor in the rat studied by isotope techniques. J Pharmacol Exp Ther 138:1-40, Oct 1962

19. Natelson EA, Blumenthol BJ, Fred HL: Acute mercury vapor poisoning in the home. Chest 59:677-678, Jun 1971

Testifying in a Hearing Loss Case

WHEN I HAVE TESTIFIED as to the noise exposure [in a hearing loss case] . . . it has always been discounted when I have taken a history or the nurse has taken it, either because it is not accurate or the patient can say he did not understand it and it was not recorded right. So now I have three forms: one is a history of occupational noise exposure, the second one is about nonoccupational noise exposure, and the third is about military noise exposure. The individual patient fills these out himself as to the time he was employed with a certain company, the type of exposure, whether he wore ear protection or not, what he thought about his hearing and whether he had any hearing tests done during that time. . . . These stand up in court, and get you out of the position of misinterpreting what the patient said.

* * * * *

IN MY DEPOSITION, I usually make a statement along these lines: "Given this patient's history, certain facts appear evident, and this is valid if the patient is telling me the truth." Then it is up to the judges and the lawyers to determine whether the patient is telling you the truth or not.

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—MICHAEL E. GLASSCOCK, III, MD, *Nashville, Tennessee*

Extracted from *Audio-Digest Otorhinolaryngology*, Vol. 11, No. 21, in the Audio-Digest Foundation's subscription series of tape-recorded programs. For subscription information: 1577 East Chevy Chase Drive, Glendale, CA 91206.